Lead Sources, Behaviors, and Socioeconomic Factors in Relation to Blood Lead of Native American and White Children: A Community-Based Assessment of a Former Mining Area

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Lead poisoning prevention requires knowledge of lead sources and of appropriate residential lead standards. Data are severely lacking on lead sources for Native American children, many of whom live in rural areas. Further, the relation of mining waste to blood lead concentrations (BPbs) of rural children is controversial. In collaboration with the eight tribes of northeastern Oklahoma, we assessed lead sources and their effects on BPbs for rural Native American and White children living in a former mining region. Venous blood lead, residential environmental (soil, dust, paint, water), and caregiver interview (e.g., hand-to-mouth behaviors, socioeconomic conditions) data were obtained from a representative sample of 245 children 1–6 years of age. BPbs ranged from 1 to 24 µg/dL. There were no ethnic differences in BPbs ($p = 0.48$) nor any patterns of excess lead sources for Native American or White children. Multiple linear regression analyses indicated that mean soil lead, mean floor lead loading, mouthing behaviors, caregivers' education, and residence in former mining towns were all strongly associated with BPbs. Logistic regression results showed mean floor dust lead loading >10.1 µg/ft^2 (odds ratio [OR], 11.4; 95% confidence interval [CI], 3.5–37.3), and yard soil lead >165.3 mg/kg (OR, 4.1; CI, 1.3–12.4) were independently associated with BPbs ≥10 µg/dL. We also found strong interactions between soil lead and poverty ($p = 0.005$), and dust and soil sources ($p = 0.02$). Our findings indicate that soil and dust lead derived largely from mining waste pose a health hazard to Native American and White children, and that current residential dust lead standards are insufficient to adequately protect children. Moreover, our finding that poor children are especially vulnerable to lead exposures suggests that residential standards should consider interactions among socioeconomic conditions and lead sources if environmental justice is to be achieved. *Key words*: adverse effects—dust, adverse effects—soil, blood lead, child, child behavior, community health planning, environmental exposure, environmental monitoring standards, epidemiology, lead poisoning, mining, North American Indians, rural health, socioeconomic factors. *Environ Health Perspect* 110(suppl 2):221–231 (2002).
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Despite dramatic reductions in environmental sources of lead and in average blood lead concentrations (BPbs) of young children in the United States since the late 1970s, lead exposure is still a major environmental health problem for U.S. children (*1–4*). National population-based data show that during 1991–1994, 4.4% of U.S. children 1–5 years of age (approximately 890,000 children) had BPbs ≥10 µg/dL, and 1.3% had levels above 14 µg/dL (*3*).

There are large racial and socioeconomic inequalities in the burden of childhood lead poisoning among U.S. children. The prevalence of BPbs ≥10 µg/dL is highest among African American, Mexican American, and poor children, especially those living in housing built before 1946 (*3*). The extent to which lead poisoning is a problem for Native American children is not known, as only one previously published study examined BPbs among Native American children (*5*). Children on Medicaid represent 60% of all children 1–5 years of age with BPbs ≥10 µg/dL, and children in families with incomes at or below 130% of the federal poverty level account for 74% of affected children (*6,7*). Further, although urban children are at greatest risk for BPbs ≥10 µg/dL, 44% of affected children live in metropolitan statistical areas with populations less than 1 million (*7*). These latter children account for 88% of all affected children (*n* = 158,880) living in newer (post-1974) housing (*7*).

Lead exposure to young children can result in a range of negative health effects, the most severe being damage to the central nervous system and death (*1*). Numerous studies have shown that children with only moderately increased BPbs (10–15 µg/dL) suffer disproportionately from cognitive and neurobehavioral deficits, including lower intelligence quotient scores and diminished attention span, bilateral coordination, visual–motor control, upper-limb speed, dexterity, and fine motor skills (*8–15*). In addition, there is mounting evidence of deleterious effects associated with BPbs even below 5 µg/dL; these include hearing loss, adverse hematologic effects, dental carries, and diminished cognitive and academic skills (*16–21*). Moreover,

lead exposure is cumulative and its effects appear to be irreversible (*9*).

There are many sources of lead in children's environments (*22*). Although lead-based paint is considered the primary lead source for U.S. children, mining and smelting waste is a potentially important lead source for children living in many small towns and rural areas throughout the United States (*22,23*). Several studies of former mining communities have found soil and dust lead to be strongly associated with BPbs in area children (*24–33*). Other studies, however, indicate that lead from mining-contaminated soil may not always pose a risk to young children (*34–36*). Similarly, studies of the bioavailability of lead from mining-contaminated soils have generated conflicting results (*23,33,37–39*). The effects on children's BPbs of soil and dust lead derived from mining activities are likely dependent on the metal species present and particle size, as well as other factors affecting exposure dose, such as amount of grass cover, number of hours spent playing in or around contaminated soil sources, season, and frequency of children's hand-to-mouth behaviors and hand washing (*23,29,30,35,37,40,41*).

Despite site-specific variations in factors affecting lead bioavailability and exposure doses, the U.S. Environmental Protection Agency (U.S. EPA) recently sought to

determine the lowest levels of lead in soil and dust at which "across-the-board abatement on a national level could be justified" (42). The new lower residential hazard levels and postabatement clearance standards were announced in January 2001 (42). The technical analyses of the U.S. EPA were based on their mechanistic model, the Integrated Environmental Uptake and Biokinetic (IEUBK) model, as well as analyses of data from the Rochester Lead-in-Dust Study (42). Lanphear and colleagues have conducted empirical analyses suggesting that these new lower standards may be insufficient to fully address the national lead problem (43,44). For example, in the original analyses of the Rochester data, Lanphear et al. estimated that 15–25% of urban children 12–31 months of age who were exposed to floor dust lead levels exceeding the new hazard standard of 40 $\mu g/ft^2$ would have BPbs ≥10 $\mu g/dL$ (44). However, the applicability of these standards to rural communities, particularly former mining sites, is not clear.

Targeted community-specific prevention of childhood lead poisoning requires precise knowledge of local lead sources and their relative contribution to BPbs. As part of the baseline evaluation for a community-based intervention study (Tribal Efforts Against Lead Project) (45), we sought to a) investigate any differences in blood lead (BPb) and residential lead source distributions for rural Native American and White children living in a former mining region; b) examine associations between mining-derived lead sources and BPbs of area children; c) quantify levels at which soil and dust lead are associated with BPbs ≥10 $\mu g/dL$; and d) assess any interactions among lead sources, children's high-risk behaviors, socioeconomic conditions, and BPbs ≥10 $\mu g/dL$ in this population.

Materials and Methods

Study Area

Ottawa County, Oklahoma, was part of the Tri-State Mining Region of Oklahoma, Kansas, and Missouri, one of the world's largest lead and zinc mining areas from the late 1800s until 1950 (46). Commercial mining in Oklahoma continued until the early 1970s, when environmental concerns and reduced yields resulted in collapse of the industry. The legacy of these mines in northeastern Oklahoma is approximately 75 million tons of lead-contaminated mine tailings and 800 acres of former floatation ponds that were used to extract metals (47). Moreover, years of mining and the ongoing practice of selling mine tailings for construction, roadways, and fill material in residential areas have resulted in widespread soil

contamination of several communities in and around the mining area. The remaining massive surface deposits of mine tailings are unprotected from surface erosion by wind, rain, and off-road vehicle traffic, and many residents still live in close proximity to these tailings. Seventy-five percent of the land affected by the piles of mine tailings is Native American owned.

In addition to lead-contaminated soils, residential paint is a potential source of lead exposure in northeastern Oklahoma. Many area homes were built prior to 1950 and have painted wooden exteriors. Further, for many of these homes, paint deterioration is a serious problem.

The northeastern portion of Ottawa County was designated the Tar Creek Superfund site in 1984 in response to potential contamination of the Roubidoux Aquifer. In the mid-1990s, a U.S. EPA emergency response team sampled soils from over 2,000 residences in the Tar Creek Superfund site. A total of 65% of sampled homes had soil lead concentrations >500 ppm, the action level determined by the U.S. EPA for this site (48). The U.S. EPA began excavating residential yard soils in the Superfund area in mid-1996. As of 30 June 1997, soil remediation had been completed on 170 homes. Prior to our study, the extent of soil contamination in towns surrounding the Superfund area had not been thoroughly investigated.

University–Tribal Partnership

The Tribal Efforts Against Lead (TEAL) Project is a collaboration among three university researchers (authors MCK, LHM, RAL) and the eight tribes of northeastern Oklahoma (Eastern Shawnee Tribe, Miami Tribe, Modoc Tribe, Ottawa Tribe, Peoria Tribe, Quapaw Tribe, Seneca–Cayuga Tribe, and Wyandotte Nation) to address the local environmental lead problem (45). Aims of this community-based intervention study are to a) reduce BPbs in Native American children in the study area; b) induce sustainable behavior change to reduce lead exposure and lead absorption in Native American children; and c) enhance the capacity of the Native American community to minimize local environmental lead exposures. The comparison population comprises non-Hispanic, White families who live in the study region. The intervention is based on a lay health advisor model and involved training 40 natural helpers from the eight tribes to educate their families, friends, neighbors, co-workers, and tribal members on lead poisoning and ways to reduce exposure to lead. Data presented here are from the baseline assessment conducted in summer and fall of 1997.

To facilitate collaborative decision making among researchers, tribes, and community

organizations, a community advisory board was formed in the first year of the project. Advisory board members included representatives from each of the eight tribes, as well as representatives from the Indian Health Service, Ottawa County Health Department, and the LEAD Agency (a local activist organization). The board met several times to provide guidance and make decisions on a range of issues related to the baseline assessment, including definition of sampling frames and screening methodology, recruitment of staff and study participants, and presentation and interpretation of study findings.

Study Population

The study sample consisted of a population-based, representative sample of Native American and White children 1–6 years of age residing within 31 contiguous census block groups in northeastern Ottawa County, Oklahoma. The study area includes Miami, the largest city in Ottawa County (1990 population = 13,142) as well as the five towns constituting the Tar Creek Superfund site, other nearby small towns, and unincorporated rural areas. With the exception of Miami, all residences in the study area were visited by two-person teams to identify and recruit eligible families. In Miami, city blocks were randomly selected within each block group, proportional to the estimated number of households with young children in each block group. At least three attempted visits were made to each residence to determine eligibility. If there were more than one eligible child per family, the child with the most recent birthday was selected to participate.

In collaboration with the TEAL Project's community advisory board, a child was classified as Native American if the primary caregiver described the child's race/ethnicity as American Indian/Native American, and if the caregiver considered her family part of the local American Indian community. The board decided that both criteria were important for inclusion, because the intervention focused on social networks within the local Native American community.

Using 1990 census data, we had estimated that families with eligible American Indian children would account for only approximately 4% of all area residences. Thus, our door-to-door sampling strategy was not projected to yield sufficient numbers of Native American children in Miami. Because visiting every home in Miami was not feasible, we recruited additional American Indian children through a variety of strategies, including visitation of all families living in tribal housing, actively recruiting families through tribally operated health and social service clinics, and by attending local powwows and other tribally sponsored events.

Staff visited 5,572 residences (approximately half of all residences in the study area) and identified 550 eligible families. A total of 137 caregivers refused to participate, 77 could not be interviewed after repeated attempts, and 5 children had incomplete interview or BPb data, resulting in a sample size of 331 (60.2% response rate). A nested case–control design was used for assessing the effect of residential environmental lead sources on BPb in children. Environmental assessments were conducted on all case (children with BPbs ≥10 μg/dL) residences ($n = 37$) and on a random sample ($n = 208$) of homes of children with BPbs <10 μg/dL. However, even after controlling for residence location (mining vs near mining), parental education, and household poverty level, case children were 3–4 times more likely than control children to have resided in their current residence for 1 month or less. Because it was possible that cases were more likely to have recently moved due to known lead sources at their previous residence, and because the current residence was unlikely to be the source of a child's current BPb, we excluded from all analyses those children ($n = 21$) who had resided at their current home for 1 month or less. Thus, the final sample size for all analyses in this paper was 224, consisting of data from 26 case and 198 control children.

Data Collection

Qualified local American Indian and White residents were hired to work as phlebotomists, interviewers, canvassers, and project coordinators for the baseline assessment.

Protection of study participants. The study protocol was reviewed by the Institutional Review Board of the University of Oklahoma Health Sciences Center. Signed consent was obtained from each participating primary caregiver prior to any data collection. Caregivers received a $15 gift certificate to a local store, and each participating child received a hand-washing kit.

Blood lead. A certified phlebotomist experienced in child venipuncture collected a venous blood sample from each participating child using a 3-cc syringe and either a 23-gauge 1-inch needle or a 25-gauge butterfly. Blood samples were kept cool and shipped daily to the Oklahoma State Department of Health laboratory for analysis by graphite furnace atomic absorption spectrometry. The state lab followed a Certified Laboratory Integrity Act proficiency plan and used reference samples approved by the Centers for Disease Control and Prevention. The detection limit was 1 μg/dL and results were reported to the nearest integer.

Interview data. Behavioral, socioeconomic, and demographic data were collected via in-home interviews with the primary caregivers of participating children. All interviews were conducted by trained interviewers and lasted approximately 1 hr. Questions on hand-to-mouth behaviors included whether the study child regularly sucked his/her thumb or fingers; put dirt, gravel, sand, or clay in his/her mouth in the past month; and put objects such as toys, rocks, gravel, sticks, pencils, or crayons in his/her mouth at least once a week. Immediately after leaving each participant's home, the interviewing team rated the cleanliness of the child's hands and face using a four-point Likert scale. Socioeconomic data included the family's total monthly take-home income and the number of adults and children supported by this income, as well as the highest education level of the primary caregiver and, if applicable, the caregiver's spouse or live-in partner.

Environmental assessments. Environmental assessments were conducted following protocols of the U.S. Department of Housing and Urban Development (HUD) and the U.S. EPA. Because of licensing requirements, we hired outside contractors to perform the environmental assessments rather than training local residents. Paint was measured *in situ* using a portable X-ray fluorescence lead paint analyzer (NITON Model XL-309; NITON Corp., Billerica, MA). All exterior and interior painted areas were analyzed for lead levels, including walls, ceilings, door and window components, siding, and soffits, as well as floors in living areas, bedrooms, bathrooms, kitchens, and playrooms. Lead was measured at the center of each painted area, with an average of 30 interior and 6 exterior surface measurements per residence. Paint condition was recorded for each painted area with a positive (≥1.0 mg/cm^2) lead reading, including estimated size of the painted area (square feet) and percentage of deterioration.

Two 1-L polyethylene labeled bottles were given to residents along with instructions for their use in collecting water samples. Residents were asked to fill one bottle in the morning from the kitchen tap before any other water was used and to fill the second after the water had been running for at least 3 min. Samples were recovered the day that they were filled and acidified to pH <2 with nitric acid.

Soil samples were collected from the front and back yards, driveway (if unpaved), dripline, and identifiable play areas. A 1-inch core sample was taken from five spots in each area and composited into a single sample. In accordance with 1995 HUD guidelines (*49*), soil samples were desiccated and processed through a 2-mm sieve prior to microwave digestion and analysis.

Using baby wipes, floor and window sill (if present) dust samples were collected from all areas of the home, including entryway, hallway, living room, play room, bathroom, kitchen, children's bedrooms, and other rooms. Floor dust was collected within the perimeter of a 1-ft^2 template taped to floors. A representative area on window sills was bounded by masking tape, and dimensions for each wipe were recorded. Samples were stored in 100 mL Whirlpaks (Nasco, Fort Atkinson, WI). Wipe samples were digested whole.

All samples were delivered to a laboratory certified by the National Lead Laboratory Accreditation Program for analysis by atomic absorption spectroscopy. Samples were analyzed according to the following methods: soil (U.S. EPA nos. 3051/7420) (*50,51*); dust (National Institute for Occupational Safety and Health method 9100) (*52*); and water (U.S. EPA no. 200.9) (*53*). The lower quantitation limits for samples were dust, 2.5 μg/ft^2; soil, 12–56 mg/kg; and water, 6 μg/L.

Statistical Analyses

Dependent variable. BPb was the main dependent variable under study. For the first set of analyses, BPb was assessed as a continuous variable. For the second set, BPb was dichotomized into levels ≥10 μg/dL and those < 10 μg/dL.

Residential lead sources. Paint indices were developed that incorporated information on the concentration of lead in the painted surface as well as the assessed size and condition of the sampled area. Paint condition was recorded on a six-point scale, indicating 0, 1–10, 11–25, 26–50, 51–75, or >75% deterioration; a corresponding deterioration value (0.01, 0.05, 0.18, 0.38, 0.63, or 0.88) was assigned to each of the six values. Paint index values were calculated as follows: *Index Value = (Pb Concentration) × (Size of Sample Area) × (Deterioration Value).* Individual paint index values were summed for each area to produce indices for bathroom, kitchen, participating child's bedroom, living room, other living areas, exterior siding, porch, total interior surfaces, and total exterior surfaces. Because exterior leaded paint may contaminate dripline soils, mean soil values were computed excluding dripline samples. All lead sources were first examined as continuous variables and then categorized into quartiles on the basis of the distributions among control (BPbs <10 μg/dL) children. To ensure adequate cell sizes for categorical analyses, the bottom three quartiles were combined to form the reference group and were compared with the highest lead quartile for each source.

Socioeconomic and behavioral measures. Interview data were entered into an EpiInfo Version 6.04C database (*54*) and validated to minimize errors. An index was created to

quantify children's hand-to-mouth behaviors. The three mouthing behaviors described above were each categorized as yes (1) or no (0) variables and then summed to generate an index ranging in value from 0 to 3. Small cell sizes precluded analysis of children with 0 behaviors, so those with 0 or 1 were combined to represent the reference group. Each child's hygiene rating was also dichotomized: children rated less than "very clean" were compared with those rated as "very clean." Family income data were used to compute the percentage of the 1997 federal poverty level; children living in families below 100% of the poverty level were compared with those living at or above poverty.

Table 1. Characteristics of children in study sample (n = 224).

Characteristic	Value
BPb (µg/dL)	
Mean	5.8
Standard deviation	3.6
Age	
Mean	3.4
Standard deviation	1.7
Race/ethnicity (%)	
Native American	42.4
White	57.6
Sex (%)	
Male	52.2
Female	47.8
Location (%)	
Near mining town	78.6
Mining	21.4
Federal poverty level (%)	
At/above poverty (≥100%)	48.2
Below poverty (<100%)	51.9
Caregivers' education[a] (%)	
>High school graduate	32.1
High school graduate	59.4
<High school graduate	8.5
Hand-to-mouth behaviors[b] (%)	
≤1	71.0
2	21.0
3	8.0
Hygiene rating[c] (%)	
Very clean	56.2
<Very clean	43.8

[a]If two caregivers, based on highest education of either caregiver. [b]Based on sum of three hand-to-mouth behaviors: sucking thumb/fingers, putting dirt in mouth, putting objects in mouth. [c]Hands and face of child rated by interviewers.

The highest educational attainment of each child's caregiver(s) was categorized for analyses; children having no caregiver with a high school degree were compared with those having at least one caregiver who had graduated from high school or completed a GED.

Statistical techniques. Except where noted, Statistical Analysis Software (55) was used for analyses. All reported *p* values are two-sided. We examined ethnic differences in distributions of BPb and lead sources using the nonparametric Wilcoxon rank sum test. Nonparametric Spearman rank correlations were computed to assess univariate associations between lead sources and BPb. Multiple linear regression modeling was used to examine the independent contributions of lead sources, behaviors, and socioeconomic variables to BPb variability. The final model produced the highest R^2 value and included only terms that remained significant at an alpha of 0.05.

Wilcoxon rank sum tests assessed associations between lead sources (as continuous variables) and elevated (≥10 µg/dL) BPbs. Unadjusted ORs and 95% CIs estimated the magnitude of associations between categorical variables and elevated BPbs in study children. Interactions among study variables were assessed via stratified analyses and the chi-square test for homogeneity of the ORs. When cell sizes were five or less, exact confidence limits were computed using EpiInfo (56). We developed a final logistic regression model, using stepwise modeling techniques, to measure the independent effects of lead sources, behaviors, and socioeconomic conditions on elevated BPbs.

Results

Children's Characteristics

BPbs of sampled children ranged from 1 to 24 µg/dL, with a mean of 5.8 (Table 1). The mean age of children was 3.4 years, with roughly equal representation of boys and girls and slightly more White than Native American children. A total of 21.4% of children lived in a former mining town and

78.6% lived in nearby towns. Approximately half (51.9%) of children lived below the federal poverty level, and though 32.1% had at least one caregiver who had some education beyond high school, 8.5% had no caregiver with a high school degree. Twenty-one percent of children engaged in two hand-to-mouth behaviors, 8.0% engaged in all three behaviors, and 43.8% were rated as having less than "very clean" hygiene.

Lead Sources and Blood Lead by Ethnicity

Table 2 presents BPb and lead source distributions for Native American and White children. Neither BPb nor log transformations of BPb were normally distributed in our sample. Soil and dust were common environmental sources of lead in homes of sampled children. Lead-based paint was a somewhat less common lead source, occurring in approximately half of sampled homes. Fewer than 10% of Native American or White homes had detectable levels of lead in their water.

There were no differences in BPb by race/ethnicity (*p* = 0.483), and there were few significant ethnic differences in environmental lead sources. Homes of White children had higher mean soil lead values than homes of Native American children (*p* = 0.033). In addition, mean sill dust lead loading values were significantly higher for White than for Native American children's homes (*p* = 0.027). However, there was no consistent pattern of excess lead sources for either ethnic group.

Associations with Blood Lead

Spearman correlations between major lead sources and BPb are shown in Table 3. BPb was most strongly correlated with mean floor dust lead loading (*r* = 0.34) and with soil lead, especially front yard soil (*r* = 0.32) and mean soil (*r* = 0.32). Interior and exterior lead-based paint were weakly correlated with BPb. Lead in water was not correlated with BPb in our sample (*p* = 0.920).

We performed multiple linear regression analyses to examine multivariate associations

Table 2. Distribution of BPbs and lead sources for Native American and White children 1–6 years of age.

| Lead source | Native Americans | | | | | | | Whites | | | | | | | Wilcoxon |
	Min	25th	50th	75th	90th	95th	Max	Min	25th	50th	75th	90th	95th	Max	p-value
BPb	1.0	4.0	5.0	7.0	9.0	13.0	19.0	1.0	3.0	5.0	7.0	11.0	14.0	24.0	0.483
Mean soil[a,b]	8.5	45.1	103.0	200.8	308.6	381.5	866.2	6.9	77.5	147.7	256.1	341.2	436.1	944.6	0.033
Front yard soil[b]	7.1	31.3	74.9	182.1	320.3	380.6	1,076.9	6.1	50.0	97.5	202.8	365.3	481.9	778.1	0.099
Back yard soil[b]	7.4	28.9	76.0	187.5	323.0	366.1	866.2	5.8	40.4	91.2	174.5	322.3	388.2	1320.1	0.466
Mean sill dust[c]	1.3	10.2	22.3	66.9	271.0	2,253.7	11,203.7	1.3	15.7	47.5	127.7	271.0	669.0	10,244.0	0.027
Mean floor dust[c]	1.3	4.1	6.1	13.7	54.5	172.0	881.2	1.3	3.3	5.8	10.3	30.2	140.8	924.0	0.151
Child's bedroom floor dust[c]	1.3	1.3	5.0	11.5	36.0	86.0	1451.0	1.3	1.3	3.5	8.0	17.0	35.5	490.0	0.070
Exterior paint index[d]	0.0	0.0	0.0	158.8	1,041.9	4,655.1	45,243.0	0.0	0.0	0.0	170.9	2,257.6	7,351.0	28,984.0	0.508
Interior paint index[d]	0.0	0.0	0.0	2.0	38.4	202.8	1,849.0	0.0	0.0	0.0	0.9	18.0	83.0	435.7	0.895
Water[e]	0.0	0.0	0.0	0.0	0.0	7.3	11.2	0.0	0.0	0.0	0.0	0.0	7.7	176.3	0.831

Abbreviations: Min, minimum; Max, maximum.
[a]Excludes dripline soil values. [b]Units are mg/kg. [c]Dust lead loading in µg/ft². [d]Based on sum of (lead-based paint value [mg/cm²] × area × deterioration value). [e]First-draw sample; units are µg/L.

of behaviors, lead sources, and socioeconomic variables with BPb (Table 4). Even when examined separately, interior paint accounted for only 1.1% of the variance in BPbs and was not significantly associated with BPb in a regression model ($p = 0.12$). The R^2 for the final model was 0.34 and included seven variables: two lead sources (mean soil lead and mean floor dust lead loading), residence location (mining vs near mining town), two behavioral variables (hand-to-mouth behaviors and hygiene rating), and two socioeconomic variables (percentage of federal poverty level and caregivers' education). Mean soil lead explained 10.1% of the variance in BPbs; location explained an additional 7.1%.

Mean floor dust lead accounted for another 3.0% of the variance, and the two behavioral variables explained an added 10.3%.

In the final model, one-unit changes in the natural logs of mean soil lead (e.g., from 50.0 to 135.6 mg/kg, or from 100.0 to 273.1 mg/kg) and mean floor lead loading (e.g., from 5.0 to 13.6 µg/ft²) were associated with 0.74 µg/dL and 0.45 µg/dL increases in BPb, respectively. A one-unit increase in the number of hand-to-mouth behaviors was associated with a 1.2 µg/dL increase in BPb. Even after controlling for lead sources and behaviors, living in former mining towns resulted in a 2.0 µg/dL increase in BPb. Having a caregiver who did not graduate from high school was associated with a 1.7 µg/dL increase, and poverty was associated with a 1.0 µg/dL increase in BPb.

Associations with Elevated Blood Lead Levels

Table 5 presents distributions of lead sources for case (BPbs ≥10 µg/dL) compared with control (BPbs <10 µg/dL) children. With the exception of exterior paint and water, values of all other lead sources were significantly higher among cases than among controls. Mean floor dust and front yard soil sources were most strongly associated with elevated (≥10 µg/dL) BPbs. Half of children with elevated BPbs had mean floor dust lead loading values below 14.5 µg/ft², compared with a median value of 5.4 µg/ft² among controls ($p < 0.0001$). The 50th percentile of mean front yard soil values was 280.1 mg/kg for cases versus 83.2 mg/kg for controls ($p < 0.0001$).

We divided each lead source into quartiles on the basis of the distributions among control children. The highest quartile was then compared with the bottom three quartiles. Table 6 shows the cutpoints used to define the highest quartiles and the exposure distributions among case and control children, as well as unadjusted and adjusted odds ratios of associations with elevated BPbs. In univariate analyses, children living in homes with the highest quartiles of lead in soil, dust, or interior paint had 2.6- to 6.7-fold increased odds of BPbs ≥10 µg/dL compared with children living in homes with lower lead exposures. Nearly 75% of children with elevated BPbs lived in homes with mean floor dust lead loading >10.1 µg/ft² or mean soil lead >197.6 mg/kg, compared with approximately 25% of control children. There were also strong univariate associations between BPbs ≥10 µg/dL and living in a mining town, hand-to-mouth behaviors, lower hygiene rating, and very young age (1 vs. ≥2 years). In addition, living below the federal poverty level and having no caregivers with a high school degree were each associated with elevated BPbs in unadjusted analyses.

The final logistic regression model included five variables (Table 6): two lead

Table 3. Correlations between lead sources and BPbs among Native American and White children 1–6 years of age.

Lead source	n	Spearman correlation coefficient	p-Value
Mean soil[a,b]	224	0.32	<0.001
Front yard soil[b]	222	0.32	<0.001
Back yard soil[b]	214	0.27	<0.001
Mean sill dust[c]	210	0.19	0.005
Mean floor dust[c]	224	0.34	<0.001
Child's bedroom floor dust[c]	224	0.24	<0.001
Exterior paint index[d]	224	0.12	0.080
Interior paint index[d]	224	0.13	0.051
Water[e]	213	−0.01	0.920

[a]Excludes dripline soil values. [b]Units are mg/kg. [c]Dust lead loading in µg/ft². [d]Based on sum of (lead-based paint value [mg/cm²] × area × percent deterioration). [e]First-draw sample; units are µg/L.

Table 4. Multiple linear regression model[a] of lead sources, behaviors, and socioeconomic variables in relation to BPb among Native American and White children 1–6 years of age.

Variable	β	SE	p-Value
Mean soil lead (mg/kg)[b,c]	0.74	0.229	0.002
Mean floor dust lead loading (µg/ft²)[c]	0.45	0.194	0.022
Location[d]	2.02	0.512	<0.001
Hand-to-mouth behaviors[e]	1.24	0.336	<0.001
Hygiene rating[f]	1.09	0.471	0.022
Federal poverty level[g]	1.02	0.451	0.025
Caregivers' education[h]	1.67	0.780	0.034

[a]R^2 is 0.34. [b]Excludes dripline soil values. [c]Regression coefficient represents change in BPb associated with a one-unit change in natural log of lead source. [d]Regression coefficient represents change in BPb associated with living in mining versus near mining towns. [e]Based sum of three hand-to-mouth behaviors: sucking thumb/fingers, putting dirt in mouth, putting objects in mouth; modeled as discrete variable: ≤1, 2, 3. [f]Dichotomous variable; hands and face of child rated by interviewers as less than very clean versus very clean (reference). [g]Regression coefficient represents change in BPb associated with living below versus at/above federal poverty level (reference). [h]Regression coefficient represents change in BPb associated with caregiver not graduating versus graduating from high school; if two caregivers, based on highest education of either caregiver.

Table 5. Distribution of lead sources by BPb levels among Native American and White children 1–6 years of age.

Lead source	≥ 10 µg/dL							< 10 µg/dL							Wilcoxon
	Min	25th	50th	75th	90th	95th	Max	Min	25th	50th	75th	90th	95th	Max	p-value
Mean soil[a,b]	18.0	106.1	261.8	363.6	759.4	800.3	944.6	6.9	58.0	118.4	197.6	300.5	379.7	866.2	0.0002
Front yard soil[b]	8.9	113.4	280.1	380.6	611.0	740.6	1,076.9	6.1	37.2	83.2	165.3	289.0	419.8	778.1	<0.0001
Back yard soil[b]	5.8	86.7	192.8	279.5	418.7	443.8	453.7	7.4	30.3	78.3	166.2	307.1	366.5	1,320.1	0.0050
Mean sill dust[c]	6.7	52.3	107.9	226.0	669.0	714.5	4303.0	1.3	13.2	27.0	80.6	249.0	772.5	11,203.7	0.0002
Mean floor dust[c]	4.5	7.9	14.5	17.9	96.0	120.2	670.0	1.3	3.3	5.4	10.1	37.5	158.3	924.0	<0.0001
Child's bedroom floor dust[c]	1.3	4.5	11.0	17.5	52.0	102.0	180.0	1.3	1.3	3.5	8.0	18.5	60.5	1451.0	0.0002
Exterior paint index[d]	0.0	0.0	86.4	401.0	8,938.4	25,530.0	28,984.0	0.0	0.0	0.0	145.0	1,045.0	4,356.0	45,243.0	0.0595
Interior paint index[d]	0.0	0.0	0.2	15.1	110.6	789.8	1186.0	0.0	0.0	0.0	0.8	18.4	125.7	1,849.0	0.0037
Water[e]	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.7	176.3	0.1453

[a]Excludes dripline soil values. [b]Units are mg/kg. [c]Dust lead loading in µg/ft². [d]Based on sum of (lead-based paint value [mg/cm²] × area × percent deterioration value). [e]First-draw sample; units are µg/L.

sources (floor dust and front yard soil), location of residence, hand-to-mouth behaviors, and caregivers' education. The point estimates for the adjusted associations tended to be substantially higher than the unadjusted associations; however, the adjusted odds ratios were less precise than the univariate associations because of the small number of cases. Although interior paint index was significantly associated with elevated BPbs in univariate analyses, it was not significant ($p = 0.16$) when added to the final multivariate model. This lack of significance, however, was likely due to small cell sizes rather than confounding by other variables (adjusted OR, 2.4; 95% CI, 0.7–8.0).

In the final model, floor dust lead loading was the strongest environmental predictor of elevated BPbs. Children living in homes with mean floor dust lead loading greater than 10.1 µg/ft² had an 11.4-fold (95% CI, 3.5–37.3) increased odds of BPbs ≥10 µg/dL compared with children living in homes with lower dust lead loading. Soil was also a significant lead source: front yard soil lead values in excess of 165.3 mg/kg were associated with a 4.1-fold (95% CI, 1.3–12.4) increased odds of elevated BPbs in children. Even after controlling for residential lead sources and socioeconomic factors, children living in a former mining town had a 5.6-fold (95% CI, 1.8–17.8) increased

odds of elevated BPbs compared with children living in nearby towns. Mouthing behaviors were also very strongly associated with BPbs ≥10 µg/dL in the final model. Children with two hand-to-mouth behaviors had a 7.0-fold (95% CI, 3.0–16.5) and those with three behaviors had a 48.9-fold (95% CI, 8.7–272.7) increased odds of elevated BPbs compared with children having one or no mouthing behaviors. In addition, the strong association between caregivers' lesser education and elevated BPbs (OR, 7.3; 95% CI, 1.4–38.4) persisted after controlling for lead sources, location, and hand-to-mouth behaviors.

We investigated whether our observed associations of high (>165.3 mg/kg) front yard soil lead and of high (>10.1 µg/ft²) mean floor dust lead with elevated BPbs persisted when homes with values above the new U.S. EPA residential hazard standards (i.e., 40 µg/ft² for floor dust lead and 400 ppm for bare soil in children's play areas) were eliminated from the final logistic regression model (42). A total of 8 cases and 27 controls had values above the standards and were removed from these analyses. In the new final model, mean floor dust lead loading remained significantly associated with BPbs ≥10 µg/dL (OR, 18.9; 95% CI, 4.3–84.1), but the association between front yard soil lead and elevated BPbs was somewhat attenuated and was no longer statistically significant (OR, 3.1; 95% CI, 0.8–12.1).

Interactions among Study Variables

Although small cell sizes prevented us from including interaction terms in the final multivariate models, we performed stratified analyses to examine interactions among study variables. There were significant interactions between soil lead and poverty ($p = 0.005$) and between dust and soil lead sources ($p = 0.023$), and borderline significant interactions between lead sources and behaviors (Table 7). For the most part, these interactions followed a similar pattern: there were no or minimal increased odds of elevated BPbs associated with a single exposure, but there were very strong associations when two exposures were present. For example, compared with nonpoor children with low (≤165.3 mg/kg) front yard soil lead, poor children with low soil lead and nonpoor children with high soil lead had no increased odds of BPbs ≥10 µg/dL; however, children who were both poor and had high yard soil lead had a 7.8-fold (95% CI, 2.5–27.1) increased odds of elevated BPbs. These differences occurred even though the distributions of yard soil lead values above the cutpoint (165.3 mg/kg) were nearly identical for poor and nonpoor children ($p = 0.78$). Likewise, compared with children

Table 6. Unadjusted and adjusted associations of lead sources, behaviors, and socioeconomic variables with elevated (≥10 µg/dL) blood lead levels in Native American and White children 1–6 years of age.

Variable	≥10 µg/dL %[a]	< 10 µg/dL %[a]	Unadjusted OR (CI)	Adjusted OR[b] (CI)
Mean soil lead (mg/kg)[c]				
Q1–3 (≤197.6)	30.8	74.7	1.0	NA
Q4 (>197.6)	69.2	25.3	6.7 (2.7–16.3)	
Front yard soil lead (mg/kg)				
Q1–3 (≤165.3)	38.5	75.0	1.0	1.0
Q4 (>165.3)	61.5	25.0	4.8 (2.0–11.3)	4.1 (1.3–12.4)
Back yard soil lead (mg/kg)				
Q1–3 (≤166.2)	45.8	75.3	1.0	NA
Q4 (>166.2)	54.2	24.7	3.6 (1.5–8.6)	
Mean sill dust lead loading (µg/ft²)				
Q1–3 (≤80.6)	47.8	75.0	1.0	NA
Q4 (>80.6)	52.2	25.0	3.3 (1.4–7.9)	
Mean floor dust lead loading (µg/ft²)				
Q1–3 (≤10.1)	30.8	74.7	1.0	1.0
Q4 (>10.1)	69.2	25.3	6.7 (2.7–16.3)	11.4 (3.5–37.3)
Child's bedroom floor dust lead loading (µg/ft²)				
Q1–3 (≤8.0)	34.6	75.8	1.0	NA
Q4 (>8.0)	65.4	24.2	5.9 (2.5–14.1)	
Exterior lead-based paint index[d]				
Q1–3 (≤145.0)	65.4	75.3	1.0	NA
Q4 (>145.0)	34.6	24.7	1.6 (0.7–3.8)	
Interior lead-based paint index[d]				
Q1–3 (≤0.75)	53.8	75.3	1.0	NA
Q4 (>0.75)	46.2	24.7	2.6 (1.1–6.0)	
Location				
Near mining town	53.8	81.8	1.0	1.0
Mining town	46.2	18.2	3.9 (1.6–9.0)	5.6 (1.8–17.8)
Hand-to-mouth behaviors[e]				
≤1	38.5	75.3	1.0	1.0
2	30.8	19.7	3.1 (1.1–8.3)	7.0 (3.0–16.5)
3	30.8	5.1	11.9 (3.9–36.9)	48.9 (8.7–272.7)
Hygiene rating[f]				
Very clean	28.0	59.8	1.0	NA
<Very clean	72.0	40.2	3.8 (1.5–9.6)	
Age, years				
≥2	65.4	84.3	1.0	NA
1	34.6	15.7	2.9 (1.2–7.0)	
Federal poverty level				
≥100%	26.9	51.1	1.0	NA
<100%	73.1	48.9	2.8 (1.1–7.0)	
Caregivers' education[g]				
≥High school graduate	80.8	92.9	1.0	1.0
<High school graduate	19.2	7.1	3.1 (1.0–9.6)	7.2 (1.4–38.4)

Q, quartile; NA, not applicable. [a]Column percentages. [b]Final logistic regression model included only those variables listed in column. [c]Excludes dripline soil values. [d]Based on sum of (lead-based paint value [mg/cm²] × area × deterioration value). [e]Based on sum of three hand-to-mouth behaviors: sucking thumb/fingers, putting dirt in mouth, putting objects in mouth. [f]Hands and face of child rated by interviewers as less than very clean versus very clean. [g]If two caregivers, based on highest education of either caregiver.

living in homes with low (≤10.1 μg/ft²) floor dust lead loading and limited mouthing behaviors, there were little increased odds of elevated BPbs for children with two or more mouthing behaviors if those children lived in homes with low dust lead (OR, 2.6; 95% CI, 0.5–14.6). Likewise, there was a only moderate association with BPbs ≥10 μg/dL for children with limited mouthing behaviors if they lived in homes with high (>10.1 μg/ft²) dust lead (OR, 3.8; 95% CI, 0.9–19.2). However, 46.2% of the case children compared with only 4.0% of control children were exposed to both high dust lead and frequent mouthing behaviors (OR, 40.1; 95% CI, 9.0–199.3).

A different pattern emerged with the interaction between floor dust lead loading and front yard soil lead. Compared with children living in homes with low (≤10.1 μg/ft²) floor dust lead loading and low (≤165.3 mg/kg) front yard soil lead, those who lived in homes with either high floor dust lead or high soil lead had similarly large increased odds of elevated BPbs as did those who lived in homes with both high yard soil and high floor dust lead. When these analyses were further subsetted to homes with low interior lead-based paint exposures, mean floor lead values ≤ 40 μg/ft², and front yard soil lead ≤ 400 mg/kg, the column percentages were similar and the interaction between dust and soil lead remained significant ($p = 0.038$).

Discussion

Ethnic Differences

Our study is the first to examine ethnic differences in BPb and in residential environmental lead sources in a community sample of Native American and White children. We observed no differences in mean BPbs of Native American compared with White children. We also found no consistent pattern of excess lead sources for either racial/ethnic group. To our knowledge, the only other community study of BPbs in American Indian children was conducted on the Navajo reservation in Arizona (5). Kazal et al. found that 2.2% of Navajo children had BPbs ≥10 μg/dL. These findings are in contrast to observations of racial differences in BPb and in the prevalence of lead sources within primarily urban populations (3,44,57–59). The higher BPbs observed among urban African American compared with White children have been shown to be due to increased levels of lead-contaminated house dust and to poorer housing conditions in the homes of African American children (60). As a majority of Native American children live in rural areas, they do not tend to have exposures to the high levels of deteriorating lead-based paint found in the older homes predominant in northeastern U.S. cities. However, our data indicate that both Native American and White children living in or near mining communities are at increased risk of BPbs ≥10 μg/dL due to mining waste.

Mining Lead Sources in Relation to Blood Lead

We investigated whether exposures to mining waste were associated with increased BPbs of exposed children. Our data showed that mean soil lead and mean floor dust lead loading were strongly and independently associated with BPbs of Native American and White children living in a former mining region. Moreover, we observed only very weak correlations, and no independent associations, between leaded paint and children's BPb. Our finding of no association between interior leaded paint and BPb was not due to inclusion of mean floor dust lead in the linear regression model; even when modeled separately, interior paint explained only 1.1% of the variance in BPbs in our population. Thus, our results suggest that mining waste, and possibly naturally occurring lead ores, are the primary derivatives of soil and dust lead in our study community. Further, despite the fact that children living outside the central mining area had high lead levels in their soil and dust (24), we found a strong association between residence in former mining towns and BPbs after controlling for residential lead sources, hand-to-mouth behaviors, and socioeconomic conditions. In sum, our results strongly support the hypothesis that lead mining wastes increase BPbs of exposed children.

Our findings are in contrast to those of Danse et al., who analyzed soil lead and BPb data from 13 communities in which mine

Table 7. Interactions among lead sources, socioeconomic conditions, behaviors, and elevated (≥10 μg/dL) blood lead levels in Native American and White children 1–6 years of age.

Variable combinations	≥10 μg/dL %[a]	<10 μg/dL %[a]	OR (CI)[b]	χ²[c]	p-Value[c]
Front yard soil lead (mg/kg), federal poverty level					
Low (≤165.3), at/above poverty (≥100%)	23.1	38.3	1.0		
Low (≤165.3), below poverty (<100%)	15.4	36.7	0.7 (0.1–3.1)		
High (>165.3), at/above poverty (≥100%)	3.8	12.8	0.5 (0.01–4.5)		
High (>165.3), below poverty (<100%)	57.7	12.2	7.8 (2.5–27.1)	7.77	0.005
Front yard soil lead (mg/kg), putting dirt in mouth					
Low (≤165.3), no	26.9	59.2	1.0		
Low (≤165.3), yes	11.5	15.8	1.6 (0.3–7.5)		
High (>165.3), no	26.9	21.4	2.8 (0.8–9.8)		
High (>165.3), yes	34.6	3.6	21.3 (5.1–88.7)	2.72	0.099
Child's bedroom floor dust lead loading (μg/ft²), child's age (years)					
Low (≤8.0), ≥2	30.8	63.1	1.0		
Low (≤8.0), 1	3.8	12.6	0.6 (0.01–5.0)		
High (>8.0), ≥2	34.6	21.2	3.4 (1.1–10.6)		
High (>8.0), 1	30.8	3.0	20.8 (4.8–90.1)	3.77	0.052
Mean floor dust lead loading (μg/ft²), hand-to-mouth behaviors[d]					
Low (≤10.1), low (≤1)	15.4	54.0	1.0		
Low (≤10.1), high (≥2)	15.4	20.7	2.6 (0.5–14.6)		
High (>10.1), low (≤1)	23.1	21.2	3.8 (0.9–19.2)		
High (>10.1), high (≥2)	46.2	4.0	40.1 (9.0–199.3)	2.14	0.144
Mean floor dust lead loading (μg/ft²), front yard soil lead (mg/kg)					
Low (≤10.1), low (≤165.3)	7.7	61.2	1.0		
Low (≤10.1), high (>165.3)	23.1	13.8	13.3 (2.2, 138.7)		
High (>10.1), low (≤165.3)	30.8	13.8	17.8 (3.2, 176.4)		
High (>10.1), high (>165.3)	38.5	11.2	27.3 (5.1, 263.7)	5.14	0.023

[a]Column percentages. [b]Exact CI, computed using EpiInfo 6.04C (54). [c]Chi square and p-value associated with Breslow-Day test for homogeneity of the odds ratios. [d]Based on sum of three hand-to-mouth behaviors: sucking thumb/fingers, putting dirt in mouth, putting objects in mouth.

tailings or milling activities were present and concluded that lead in mine tailings is not readily bioavailable (35). For five of the six U.S. tailing sites they studied, there was no local control population; instead they considered the observed mean BPbs at these five sites to be within "normal" range based on National Health and Nutrition Examination Survey (NHANES) data. However, NHANES data are not an appropriate external comparison population for mining communities; absent mining lead sources, these rural communities would be expected to have lower mean BPbs than a national sample, as the housing stock in these communities is generally newer, with less leaded paint, and has considerably less cumulative exposure to leaded gasoline than urban areas (23,43,61). Further, to most accurately assess the effect of soil lead on BPbs, analyses should go beyond a simple comparison of mean values to examine a range of soil lead values in relation to BPbs.

Our findings are consistent with several other studies that assessed the effect of mining lead sources on BPbs of children (25–31). Most notably, Murgueytio et al. studied former mining sites in Missouri that, like our site, are part of the Tri-State Mining Region (25,26,28). At the Jasper County Superfund site in southwest Missouri, the authors found that soil lead explained 20% of the total variance in BPb, dust explained another 4%, and paint and water each explained less than 0.2% (25). Their study could not, however, distinguish mining from smelting lead sources. At the Big River Mine Tailings site in southeastern Missouri, where there were minimal smelting operations, the researchers again found that mining waste accounted for at least as much variation in children's BPbs as did other sources (26). They also concluded that mining waste was the only reasonable explanation for the observed doubling in prevalence of BPbs ≥10 µg/dL in the mining community compared with a rigorously selected control community. Our study, also based in a community with limited smelting activity, found that mean soil lead explained the largest portion (10.1%) of the variance in BPbs, followed by residential location (mining vs near mining), which explained an added 7.1%. Mean floor dust lead loading accounted for an additional 3.0% of the variance.

Residential Lead Hazard Standards

The new U.S. EPA residential hazard levels and postabatement clearance standards regard all residential dust and soil lead, regardless of lead source, to be a "lead-based paint hazard" if their condition and location "would result in adverse human health effects" (42). Under these new uniform

national standards, floor dust lead is defined as a hazard if the weighted average of all floor dust wipe samples exceeds 40 µg/ft². The new hazard standard for soil lead is 400 ppm in bare soil in children's play areas or 1,200 ppm in bare soil in the remainder of the yard. It should be noted that in our study most yards contained large areas of bare soil, and that study children used their front yards as play areas.

Our findings indicate that, at levels far below the new residential standards, exposure to floor dust lead loading is strongly associated with children's BPbs ≥10 µg/dL. After controlling for other lead sources, location, mouthing behaviors, and caregivers' education, we found that mean floor dust lead loading greater than 10.1 µg/ft² was associated with an 11.3-fold increased odds of BPbs ≥10 µg/dL in study children. When we reanalyzed our data eliminating homes with lead levels exceeding the new standards, we observed similarly strong and statistically significant associations with BPbs ≥10 µg/dL for dust lead loading >10.1 µg/ft².

Our results also suggest that mean yard soil lead values between 165.3 and 400 mg/kg may be associated with BPbs ≥10 µg/dL. We observed an adjusted 4.1-fold increased odds of elevated BPbs for children exposed to front yard soil lead levels greater than 165.3 mg/kg. However, when we reran these analyses excluding homes with soil lead values above 400 mg/kg and mean floor dust lead above 40 µg/ft², the observed adjusted association with yard soil lead >165.3 mg/kg was somewhat attenuated (OR, 3.1) and was no longer statistically significant. These latter analyses were limited by small cell sizes. Another limitation of our soil lead data is that we used what the U.S. EPA has termed the "total soil sample," which is the soil that remains after passing through a 2.0-mm sieve (62). The lead content of these larger particle sizes may be considerably less than the lead values of the fine fraction, which passes through a 250-µm sieve. The fine fraction is considered more representative of the particle size that a young child would ingest via hand-to-mouth behaviors. Thus, it is possible that a lead concentration of 165 mg/kg in the total soil fraction may be comparable to a value above 400 in the fine soil fraction.

The U.S. EPA analyses indicated that at the new hazard standards (e.g., floor dust levels at 40 µg/ft²), children would have roughly a 5% risk of BPbs ≥10 µg/dL (42). In contrast, in an analysis of data on urban children 12–31 months of age from Rochester, New York, that controlled for other lead sources, behaviors, and parents' education, Lanphear et al. estimated that dust lead standards would need to be set at 5 µg/ft² to achieve the U.S. EPA target risk

level of 5% (44). The recent pooled analyses of data by Lanphear et al. from 12 epidemiologic studies further supported these findings (43). In response to public comments on the proposed ruling, the U.S. EPA disputed these findings on methodologic grounds, arguing that the analyses of Lanphear et al. did not adequately address the contributions of soil lead and deteriorated leaded paint to exposure (42). Although our sample size was limited, our data support the findings of Lanphear et al. When we limited our final logistic regression model to homes with exposures at or below the new U.S. EPA residential hazard standards (i.e., 40 µg/ft² for floor dust lead and 400 ppm for bare soil in children's play areas), we still found a very strong association (OR, 18.9; 95% CI, 4.8, 84.1) between children's exposure to floor dust lead loadings of 10.2–39.9 µg/ft² and elevated BPbs, even after controlling for residence location, caregivers' education, mouthing behaviors, and yard soil values ≤400 mg/kg. In addition in homes (n = 142) with yard soil levels ≤400 mg/kg, mean floor dust lead loadings ≤40 µg/ft², and minimal interior lead-based paint exposures, we found that 60.0% of children with BPbs ≥10 µg/dL, in contrast with only 12.1% of controls, had mean floor dust lead loadings of 10.2–39.9 µg/ft². Together, these empirical studies suggest that the new U.S. EPA standards will be insufficient to achieve the elimination of children's exposure to the harmful effects of lead.

Behaviors, Socioeconomic Conditions, and Observed Interactions

Although elimination of lead sources is of primary importance in the prevention of childhood lead poisoning, our data show that children's hand-to-mouth behaviors and hygiene practices, as well as family socioeconomic conditions, are important contributors to children's body lead burden. All of these variables remained significant in our final multiple regression model after controlling for lead sources and residence location. In our final logistic regression analyses of elevated BPbs, we found that mouthing behaviors and caregivers' education level were associated with large increases in the odds of BPbs ≥10 µg/dL. These findings are consistent with several other studies of both urban and rural children (29,30,37,40,43,44,57, 59,60,63,64).

Few studies, however, have examined interactions among behaviors, socioeconomic conditions, and lead sources (43). Identifying heterogeneity in risk within populations, and in particular examining whether environmental contaminants have a disproportionate impact on certain socioeconomic or racial

groups, is crucial for the advancement of environmental justice. We observed strong and statistically significant interactions between soil lead and poverty and between dust and soil sources. The observed interaction between soil lead and poverty suggests that the entire effect of soil lead occurred among poor children, who accounted for roughly half of our sample: Among non-poor children, there were no increased odds of elevated BPbs associated with exposure to high (>165.3 mg/kg) soil lead. However, among poor children, high soil lead exposure was associated with over a 10-fold increased odds of elevated BPbs. Limited cell sizes prevented us from further exploring this interaction. It is possible that, compared with other children exposed to high soil levels, exposed poor children had exposures to higher concentrations or greater quantities of other lead sources, were exposed to such sources for a longer duration, or had other host factors, such as more frequent hand-to-mouth behaviors, poorer nutritional status, or greater periods of food insufficiency that may have increased their lead exposure or rate of lead uptake from the gastrointestinal tract.

Our data were also suggestive of interactions between behaviors and lead sources. We found that exposure to high (>165.3 mg/kg) yard soil lead was not significantly associated with elevated BPbs among children who did not put dirt in their mouths, but for children who practiced this behavior, high yard soil lead resulted in over a 10-fold increased odds of elevated (≥10 µg/dL) BPbs. Likewise, our findings showed that the association between exposure to bedroom floor dust loading >8.0 µg/ft^2 and elevated BPbs was far greater for 1-year-old children than for children 2 years of age or older. The latter interaction may explain our unadjusted association of very young age with elevated BPbs; 1-year-old children may spend more time playing or lying on their bedroom floors than even slightly older children. Our observed interactions suggest that the new U.S. EPA standards may be particularly inadequate for protecting poor children and children who engage in frequent mouthing behaviors.

Limitations

It is important to consider our study's limitations. First, we had a 60.2% response rate, so our findings may not be representative of all children in the study area. The primary reasons for parental refusal were not wanting their child to have a venous blood draw or their child having previously received a BPb test. Thus, it is possible that more parents of children at lower risk for elevated BPbs refused to participate. However, we have no indication that nonresponse among these children was biased with respect to lead

sources or other study variables of interest, so it is unlikely that nonresponse biased our main study findings. Second, we sampled children only during the summer and early fall, when exposures to lead sources are highest; our observed relations between study variables and BPbs may vary by season. Third, our sample had a limited range of BPbs, from 1 to 24 µg/dL, so we were not able to estimate associations between lead sources and higher BPbs. Fourth, as mentioned previously, the small number of children with BPbs ≥10 µg/dL in our sample limited the precision of some of our estimates as well as our ability to examine interactions in multivariate analyses. Fifth, our sample is from a historic mining region and may not be generalizable to urban settings where lead-based paint and leaded gasoline are the primary lead derivatives of dust and soil lead. A sixth limitation is that we were unable to measure nonresidential sources of lead in children's environments, such as lead sources in other homes in which children may spend time, direct exposure to playing on mine tailings, and exposure to roadways covered with mine tailings. Our findings from a previous study in the same area suggest that residential driveways covered with mine tailings can have soil lead concentrations of over 10,000 mg/kg. These sources are more prevalent in the former mining towns and may explain why residence in these towns was strongly associated with BPbs ≥10 µg/dL even after controlling for residential lead sources. Lastly, the Native American children in our sample are by no means representative of all Native American children. There is tremendous cultural and geographic diversity among U.S. Native peoples, who belong to over 500 recognized tribes and Alaskan villages as well as numerous unrecognized tribes.

Community-Based Research

This research is funded by NIEHS as part of their Community-Based Prevention/Intervention Research initiative. The goal of this initiative is to fund culturally relevant prevention and intervention research in economically disadvantaged and underserved communities negatively impacted by environmental contaminants. One intended outcome of this program is to improve the capacity of affected communities to be true partners in research, thereby maximizing the community relevance of research findings and the effectiveness of resultant health interventions.

Community participation has been crucial to the success of the TEAL Project. Our community advisory board and our data collection staff, all comprised entirely of community members, were a continual resource throughout the baseline assessment. They

defined key study variables (such as the highly culturally bound issue of defining "Native American" for study purposes), developed key recruitment strategies, reviewed survey instruments for cultural relevance and appropriateness of question wording, improved participants' comfort level with the home-based interviews and blood draws to increase response rates, helped researchers present information in understandable ways for a lay audience, and provided critical feedback on policy implications of study findings. Regarding the latter, when we first presented data to the community advisory board showing that soil and dust lead sources were prevalent in both mining and nearby towns and that the prevalence of BPbs ≥10 µg/dL was higher than expected even in towns outside the mining area (*24*), the discussion focused on the need for an ordinance to stop the continued sale and distribution of mine tailings in their communities and the need to widely distribute these findings to non-Superfund residents. They also discussed the possible need for ongoing U.S. EPA soil cleanup efforts to expand beyond the boundaries of the Superfund area.

Community residents, and the community as a whole, in turn benefited from the TEAL Project. Through their participation, board members and TEAL Project staff increased their knowledge of lead sources, lead poisoning prevention strategies, data collection methods, epidemiologic study design issues, and scientific approaches to community health concerns. This increased knowledge and skill base of individual community members enhanced community capacity to respond to the local severe environmental lead problem. The TEAL Project also created jobs for community residents and provided them with research skills that may be transferred to other job settings and community health issues.

The TEAL Project's intervention arm, the Society of Clan Mothers and Clan Fathers, targeted their lead poisoning prevention strategies on the basis of community-specific data (*45*). The use of local data was especially crucial, as lead-based paint, the focus of most national lead education interventions, was not the primary lead source in our study community. In response to our data showing the importance of dust as a lead source, the Society assisted local tribes in obtaining Hepa vacuum cleaners that could be used by area residents. In addition, Clan Mothers and Fathers educated members of their social networks about local lead sources and designed educational materials and outreach activities stressing the importance of hand washing and reducing, to the extent possible, children's hand-to-mouth behaviors. The Society's education

efforts also contributed to the decision by the City of Miami to explore regulating the sale and use of mine tailings.

Conclusion

A major advantage of conducting research in collaboration with communities is that the study findings often have immediate relevance and policy implications for the community. On a local level, our data highlighted the critical need for continued soil remediation from area residences at the Tar Creek Superfund site, as well as the need for an ordinance to restrict the sale and distribution of mine tailings.

On a national level, the complete elimination of blood lead levels ≥10 µg/dL is a health priority for 2010 (65). To meet this goal, federal, state, and local governments must focus on the removal of lead sources before children are exposed (66). Our study findings suggest that dust and soil lead derived from mining waste pose a health hazard to Native American and White children living in former mining communities. Further, our data indicate that certain groups of children, e.g., those who live in poverty, or those who engage in more frequent hand-to-mouth behaviors, are at very high risk of BPbs ≥10 µg/dL, even when exposed to environmental lead levels below the new U.S. EPA standards. Because a high percentage of U.S. children fall within these risk groups, as do a majority of children exposed to residential lead sources, our findings have important implications for the advancement of environmental justice. Our data suggest that justice will not be achieved unless interactions among socioeconomic conditions, child behaviors, and lead sources are examined in evaluations of the effectiveness of the U.S. EPA's new standards.

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